third left costal cartilage with the sternum, and conducted along the aorta. His temperature was 101.4° in the morning, and 103.2° in the evening. It had been so within a few points for four days, but the patient did not show much distress. His tongue was moist, his pulse full and strong. He was treated with a blister over the cardiac region, and digitalis. On August 4 there was some vomiting and a little intermittence of pulse. On the 5th there was some presternal pain; the pulse was full, but intermittent. The murmur was traced along the vessels of the neck. The discharge was almost *nil.* On the 6th the evening temperature again rose to 102.9°, and there was palpitation for the first time. There was abundant sweating; constipation; tongue moist. On the 8th there were some râles heard in the lungs, believed to be due to œdema. After the 13th the patient gradually improved. On the 23d there was nothing to note except that he was feeble, that he still sweated, that the murmur was scarcely altered, and that the urethral discharge had quite disappeared. On the 26th his general improvement was still maintained, but the gonorrhœa reappeared. Dr. Marty believed that the murmur was due to the occurrence of proliferating endocarditis at the level of or on one of the aortic valves.

We have only space to summarize Dr. Marty's general conclusions, which are as follows: Gonorrhœa may be complicated with inflammation of all the serous membranes, and may act in a direct way on each of them. Rheumatism is by no means a necessary middle term between the specific lesion and the lesion of the serous membrane, although the cases of coexistence of the two complications are most frequent. The cardiac complications are rare. Of the several orifices the aortic one is most commonly attacked. Endocarditis appears to have occurred as frequently as pericarditis, if not more so.—*Med. Times and Gaz.*, Dec. 23, 1876.

15. *Anomalous Conduction of Mitral Murmurs.*—At a late meeting of the Medical Society of London, Dr. Sansom related (*Med. Times and Gaz.*, Jan. 27, 1877) two cases recently under his care in the London Hospital, illustrating anomalous conduction of mitral murmurs. The patient in the first case was a man, twenty-four years of age, who had had three attacks of acute rheumatism, and had on a former occasion been a patient in the London Hospital, when a presystolic mitral murmur was noted. There was now a loud musical first-sound murmur, and a systolic thrill could be detected, whose maximum was a little to the left of the base of the ensiform cartilage. It was so strongly marked in the tricuspid area that most competent observers thought it must be due to tricuspid regurgitation. There was now no presystolic murmur. Dilatation of the right side and the usual signs of failing heart preceded death. At the autopsy the mitral orifice was found to be narrowed so as to admit only one finger, and to be surrounded by a calcareous ring. The calcareous matter was attached in largest mass to the interventricular septum. Dr. Sansom considered that this dense calcareous material had acted as a conductor of sound to the interventricular septum, and so by the right apex to the ear; hence the abnormality of position of the murmur. In the second case the patient was a man aged fifty, whose only previous illnesses were chorea at eight years of age, and scarlatina at fourteen. Lately he had had rheumatoid pains, but no fever. On auscultation, a musical presystolic murmur could be heard over an area not greater than a circle of one inch and a half in diameter, just to the left of the base of the ensiform cartilage. A little to the left of this spot there was a loud rough first-sound murmur. Twelve months after first coming under observation the presystolic murmur ceased to be audible. Before death, dilatation of the right side was manifest, with venous pulse and throbbing liver. To relieve ascites, paracentesis abdominis was performed, and it was noted that the urine, which just previously had been highly albuminous, now contained very little albumen, but was highly saccharine. The diagnosis in this case was arrived at by experience of the former. It was that there was mitral stenosis, the characteristic presystolic murmur of which had been conducted by dense material to the interventricular septum, and thence to the apex; that, considering the musical character of the murmur, the valve aperture was probably a calcareous

ring; that the murmur had ceased to be audible because the left auricle had become dilated and its muscle enfeebled; that there was, in addition, incompetence both of mitral and tricuspid valves. This diagnosis was exactly confirmed by the autopsy. Dr. Sansom thought the cases worthy of record, as the observations might be useful in diagnosis.

16. *Variations of Urea in Disease of the Liver.*—In a paper, the first part of which appears in the *Archives de Physiologie*, July, 1876, M. P. BROUARDEL gives a historical sketch of the views which have been from time to time published respecting the part taken by the liver in the formation of urea, from which it would appear that the great weight of evidence, both pathological and physiological, goes to show that a direct relation exists between the functional activity of the liver, and the excretion of urea, so that in all probability a great part of the urea which appears in the urine is formed in the liver. On account of the want of accurate clinical research upon this question, the author proposes to investigate the exact amount of urea excreted daily in various hepatic affections, and by a comparative analysis to arrive at a definite conclusion on the subject. 1. Of fatal jaundice (acute atrophy), no original cases are given; but from those recorded by Frerichs and others, it is regarded as satisfactorily proved that in proportion as this disease progresses the amount of urea steadily diminishes, and finally disappears. 2. In four cases of fatal jaundice produced experimentally in dogs by the hypodermic injection of phosphorated oil, the amount of urea excreted in twenty-four hours always diminished in direct proportion to the amount of phosphorated oil injected, and the severity of the general symptoms seemed also to vary with the amount of the poison used, and the diminution of urea. 3. In four cases of typhoid jaundice (*Ictère pseudo-grave*) with reduction of the size of the liver, the formation of the urea was lessened in the early stages of the disease, and during the typhoid it was observed that the urea was reduced to a minimum. The rather sudden disappearance of the severe symptoms (crisis), was found invariably to take place on the same day as the normal amount of urea appeared in the urine. 4. In three cases of simple spasmodic jaundice, the amount of urea seemed increased, and at the same time the size of the liver was augmented. The diminution in the amount of urea excreted in jaundice is thus made to bear a definite relation to the severity of the symptoms, and the gravity of the prognosis in any given case, and as a general rule it has been found that the formation of urea is in direct proportion to the size of the liver.

The paper is completed in the number of the same journal for September and October. M. Brouardel thus sums up the conclusions at which he has arrived, as to the variations in the excretions of urea in the different forms of liver-disease.

1. In an acute yellow atrophy, the urea diminishes or disappears entirely.

2. In phosphorus poisoning, although each dose of the drug causes a temporary augmentation of the quantity of urea excreted, it slowly diminishes.

3. Some pseudo-malignant cases of acute jaundice have the primary diminution of urea followed by a urinary crisis, and plenty of urea is secreted; the liver, which at first seemed to become smaller, recovers its ordinary size.

4. In simple jaundice, the urea is not diminished, sometimes it is augmented. The quantity of urea is a means of prognosis.

5. In hepatic abscess, according to Parkes, at first the urea is augmented, but this requires verification; as the liver-substance is destroyed it diminishes, although fever may be present.

6. In biliary calculus, with obliteration of the duct and destruction of liver-substance, the urea is diminished, especially during the attack of biliary colic. It is also diminished in hepatic intermittent fever.

7. In both the hypertrophic and the atrophic forms of cirrhosis, the urea is diminished.

8. In the congested and hardened liver of heart disease, the urea is diminished.

9. In the fatty liver of phthisis, the urea falls to low figures.